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Smoking as a Multifactorial Ototoxic Risk Factor: Associations with Elevated Hearing Thresholds, Tinnitus, and Vertigo

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ABSTRACT

Objectives: This retrospective study evaluated the association between smoking, hearing loss, tinnitus, and vertigo in 443 patients evaluated between 2015 and 2025. The primary aim was comparing Pure-Tone Audiometry (PTA) thresholds between smokers and non-smokers.

Methods: Audiological data, including PTA thresholds, tinnitus, and vertigo presence, were analyzed after excluding patients with confounding otologic factors. Group comparisons utilized independent t-tests and Two-Way ANOVA to assess main and interaction effects.

Results: Among 443 patients (218 smokers, 225 non-smokers), smokers exhibited significantly higher mean PTA thresholds in both better-hearing (22.1 vs. 18.0 dB; p<0.001) and worse-hearing ears (25.3 vs. 20.1 dB; p<0.001). Tinnitus prevalence was significantly higher in smokers (43.1%) compared to non-smokers (27.1%) (p<0.001), as was vertigo (27.5% vs. 20.4%; p=0.047). A positive correlation was observed between daily cigarette consumption and hearing thresholds.

Conclusion: Smoking is significantly associated with elevated hearing thresholds and increased prevalence of tinnitus and vertigo. These findings confirm smoking as a multifactorial risk factor for auditory and vestibular dysfunction, underscoring the critical need for smoking cessation interventions.

Keywords: Smoking, Hearing Loss, Vertigo, Tinnitus, Ototoxicity

ÖZ

Sigaranın Multifaktöriyel Bir Ototoksik Risk Faktörü Olarak Rolü: Artan İşitme Kaybı, Tinnitus ve Vertigo ile İlişkisi

Amaç: Bu retrospektif çalışma 2015 ile 2025 yılları arasında değerlendirilen 443 hastada sigara kullanımı ile işitme kaybı, tinnitus (kulak çınlaması) ve vertigo (baş dönmesi) arasındaki ilişkiyi değerlendirmiştir. Birincil amaç, sigara içenler ile içmeyenler arasındaki Saf Ses Odyometrisi (PTA) eşiklerini karşılaştırmaktır.

Yöntemler: Karıştırıcı otolojik faktörlere sahip hastalar dışlandıktan sonra; PTA eşikleri, tinnitus ve vertigo varlığını içeren odyolojik veriler analiz edilmiştir. Gruplar arası karşılaştırmalarda, ana ve etkileşim etkilerini değerlendirmek için bağımsız t-testleri ve İki Yönlü ANOVA kullanılmıştır.

Bulgular: 443 hasta (218 sigara içen, 225 içmeyen) arasında, sigara içenler hem daha iyi işiten kulakta (18,0 dB'ye karşı 22,1 dB; p<0,001) hem de daha kötü işiten kulakta (20,1 dB'ye karşı 25,3 dB; p<0,001) anlamlı derecede daha yüksek ortalama PTA eşikleri sergilemiştir. Tinnitus prevalansı sigara içenlerde (%43,1), içmeyenlere (%27,1) göre anlamlı derecede daha yüksek bulunmuştur (p<0,001); aynı durum vertigo için de geçerlidir (%27,5'e karşı %20,4; p=0,047). Günlük sigara tüketimi ile işitme eşikleri arasında pozitif bir korelasyon gözlemlenmiştir.

Sonuç: Sigara kullanımı, yükselmiş işitme eşikleri ve artmış tinnitus ile vertigo prevalansı ile anlamlı derecede ilişkilidir. Bu bulgular, sigaranın işitsel ve vestibüler (denge) disfonksiyon için çok faktörlü bir risk faktörü olduğunu doğrulamakta ve sigara bırakma müdahalelerinin kritik ihtiyacını vurgulamaktadır

Anahtar kelimeler: Sigara, İşitme kaybı, Tinnitus, Vertigo, Ototoksisite

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INTRODUCTION

It is estimated that more than 1.5 billion people worldwide experience some degree of hearing difficulty, with approximately 430 million affected by moderate or more severe loss in the better-hearing ear (WHO, 2021). Hearing loss has become a major public health issue, with increasing prevalence and broad consequences for social, mental, and physical health. It hampers speech perception, limits effective communication, and often leads to social withdrawal (Kazempour, Tajvar, & Heirani, 2023; Huang, Zhou, Chen, Du, Lin, & Ye, 2025). These effects substantially reduce quality of life and may contribute to depression, cognitive decline, and even dementia, highlighting the importance of early prevention and management.

Hearing loss may result from various environmental factors, with noise exposure, sudden idiopathic loss, ototoxic agents, and age-related decline being the most prominent contributors.

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In addition, several other conditions can also play a role in its development, including sleep disturbances (Bener, Erdogan, & Griffiths, 2024), smoking (Myszel & Skarżyński, 2024; Bhatt & Upreti, 2025; Istrate, Hasbei-Popa, Iliescu, Ghita, & Ghita, 2021), hypertension, and cardiovascular diseases (Capaccio, Ottaviani, Cuccarini et al., 2007; Einer, Tengborn, Axelsson, & Edstrom, 1994; de Oliveira Penido, Ramos, Barros, Cruz, & Toledo, 2005; Chau, Lin, Atashband, Irvine, & Westerberg, 2010). Among these, smoking has recently gained attention as a preventable and modifiable risk factor with multiple adverse effects on the auditory system. Smoking is a well-established risk factor for a wide range of systemic diseases, including cardiovascular disorders, respiratory conditions, and multiple types of cancer (Seo, Park, Kim, & Lee, 2023). Its harmful effects may also extend to the auditory system, primarily through oxidative stress and vascular alterations that compromise cochlear function (Garcia Morales, Ting, Gross et al., 2022; Cruickshanks, Klein, Klein, Wiley, Nondahl, & Tweed, 1998). Cigarette smoke contains numerous toxic substances capable of generating free radicals, which can disrupt metabolic processes within the cochlea or reduce cochlear blood flow, leading to hypoxic damage (Garcia Morales et al., 2022).

Although smoking has been investigated as a potential contributor to hearing loss (HL), findings across studies have been inconsistent. While several reports indicate a positive correlation between smoking and HL, others have failed to confirm such an association (Garcia Morales et al., 2022; Dawes, Cruickshanks, Moore, Edmondson-Jones, McCormack, Fortnum, & Munro, 2014). Biologically, nicotine and other ototoxic components in cigarette smoke may exert direct toxic effects on cochlear hair cells, or indirectly induce cochlear hypoxia through increased blood viscosity and reduced oxygen availability (Kazempour et al., 2023; Dawes et al., 2014). These mechanisms collectively may lead to irreversible damage of the sensory hair cells and subsequent impairment in auditory function.

METHODS

This retrospective study analyzed patients attending the Otorhinolaryngology and Audiology clinics at Istanbul Oncology Hospital between 2015 and 2025 to evaluate the association between smoking and hearing loss. Audiological data, including pure-tone audiometry (PTA) thresholds for both better and worse ears, as well as the presence of tinnitus or vertigo, were collected. Statistical analyses were performed using IBM Statistical Package for Social Sciences (SPSS) program. Group comparisons were conducted using independent t-tests or Mann-Whitney U tests, and categorical variables were analyzed with chi-square tests. Correlations between smoking and hearing thresholds were assessed, with $p < 0.05$ considered statistically significant. Data collection was independently performed by two researchers to minimize bias, and incomplete records were excluded.

Inclusion Criteria

Patients who met the study criteria with complete audiological records were included. Collected variables comprised age, sex, PTA thresholds for both ears, and the presence of tinnitus or vertigo. These data enabled a clear comparison of auditory function between smokers and non-smokers.

Exclusion Criteria

Patients were excluded if they had a history of ototoxic drug use, prior head and neck radiotherapy, previous ear surgery, chronic otitis media, Patients with retrocochlear lesions excluded based on magnetic resonance imaging (MRI) findings were not included in the study, or use of cancer-related or immunotherapy drugs. Incomplete or missing records were also excluded to ensure data integrity.

RESULTS

The mean age of the non-smoking group (n=225) was calculated as 33.79±10.64 years, and the mean age of the smoking group was calculated as 34.88± SD=10.46 years.

Table 1. Distribution of gender, tinnitus and vertigo according to smoking status

Variable		Non-Smokers n (%)	Smorkers n (%)
Gender	Female	105 (46.7%)	94 (43.1%)
	Male	120 (53.3%)	124 (56.9%)
Tinnitus	Absent	164 (73.2%)	126 (57.8%)
	Present	60 (26.8%)	92 (42.2%)
Vertigo	Absent	183 (81.7%)	159 (73.3%)
	Present	41 (18.3%)	58 (26.7%)

***p <0.001

Upon examining Table 1, it is observed that the demographic distributions of the smoking and non-smoking participants are similar, but significant differences exist in terms of clinical symptoms. Tinnitus was observed in 26.8% of the non-smoking group, while this rate was determined to be 42.2% in the smoking group. Similarly, the proportion of those experiencing vertigo was 18.3% among non-smokers, compared to 26.7% among smokers.

Table 2. Comparison of PTA values according to smoking status

Variable	Grup	n	Ortalama ± SS	t	p
PTA (Good ear)	Non-smokers	225	17.95±9.03	-4.63	<0.001***
	Smokers	218	22.14±10.01		
PTA (Bad ear)	Non-smokers	225	20.14±10.16	-5.13	<0.001***
	Smokers	218	25.32±11.11		

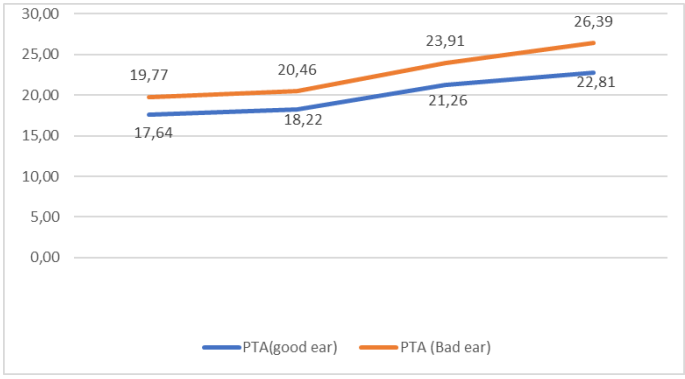

Figure 1. Comparison of PTA values based on sex in the groups.

Upon examining the table, significant differences were observed in both PTA (Good ear) [t (441)=-4.63, p <0.001] and PTA (Bad ear) [t (441)=-5.13, p <0.001] according to smoking status. These findings indicate that smokers have significantly higher mean PTA values in both the good ear (mean=22.14±10.01) and the bad ear (mean=25.32±11.11) compared to non-smokers (good ear mean=17.95±9.03; bad ear mean=20.14±10.16).

In other words, smoking negatively affects hearing thresholds, resulting in higher PTA values (i. e., worse hearing) in both ears.

Upon examining the table, no statistically significant differences were observed between males and females in mean PTA (Good ear) and PTA (Bad ear) values within both the non-smoking and smoking groups (p >0.05). Although males had slightly higher mean PTA values than females in both groups, these differences were not statistically significant. These results indicate that gender does not have a significant effect on hearing thresholds in either non-smokers or smokers.

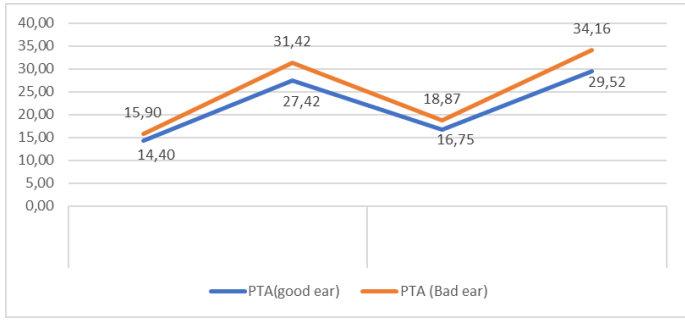

Figure 2. Comparison of PTA values based on presence of tinnitus in the groups

Upon examining the table results indicate that, in both smokers and non-smokers, individuals with tinnitus have significantly higher PTA values compared to those without tinnitus (all t-tests, p <0.001). This finding suggests that the presence of tinnitus is associated with increased hearing thresholds, i.e., hearing loss. However, smoking status appears to further amplify this relationship.

- In the non-smoking group, the mean PTA for individuals without tinnitus was approximately 14 dB for the good ear and 16 dB for the Bad ear, whereas in those with tinnitus, these values increased to 27–31 dB.
- In the smoking group, the mean PTA for individuals without tinnitus was approximately 17 dB for the good ear and 19 dB for the Bad ear, while in those with tinnitus, values rose to 29–34 dB.

These differences demonstrate that tinnitus is significantly associated with hearing loss in both smokers and non-smokers. However, because the mean differences are higher in smokers, smoking can be considered a factor that exacerbates the impact of tinnitus on hearing.

Table 3. Comparison of PTA values according to smoking status and presence of tinnitus

Smoking Group	Variable	Tinnitus	n	Mean ± SD	t	p
Non-smokers	PTA (Good ear)	Negative	164	14,40 ± 6,84	-12,46	<0.001***
		Positive	60	27,42 ± 7,15		
	PTA (Bad ear)	Negative	164	15,90 ± 7,15	-13,85	<0.001***
		Positive	60	31,42 ± 8,12		
Smokers	PTA (Good ear)	Negative	126	16,75 ± 7,19	-11,96	<0.001***
		Positive	92	29,52 ± 8,52		
	PTA (Bad ear)	Negative	126	18,87 ± 7,39	-13,70	<0.001***
		Positive	92	34,16 ± 9,08		

Table 4. Comparison of PTA values according to smoking status and presence of vertigo

Smokers Group	Variable	Vertigo	n	Mean ± SD	t	p
Non-smokers	PTA (Good ear)	Negative	183	16.00 ± 8.10	-8.01	<0.001***
		Positive	41	27.00 ± 7.20		
	PTA (Bad ear)	Negative	183	17.64 ± 8.62	-9.47	<0.001***
		Positive	41	31.66 ± 8.31		
Smokers	PTA (Good ear)	Negative	159	19.94 ± 9.27	-5.75	<0.001***
		Positive	58	28.19 ± 9.63		
	PTA (Bad ear)	Negative	159	22.12 ± 9.66	-7.98	<0.001***
		Positive	58	34.12 ± 10.17		

Upon examining the table, significant differences in hearing levels were observed according to the presence of vertigo in both non-smoking and smoking participants (t-values ranging from -5.75 to -9.47, $p < 0.001$).

In the non-smoking group, participants with vertigo had markedly higher mean hearing thresholds for both the good ear (Mean=27.00±7.20) and the bad ear (Mean=31.66±8.31) compared to those without vertigo (Good ear Mean=16.00±8.10; Bad ear Mean=17.64±8.62). These differences were statistically significant (Good ear: $t(222)=-8.01$, $p < 0.001$; bad ear: $t(222)=-9.47$, $p < 0.001$). This finding indicates that individuals exhibiting vertigo have significantly elevated levels of hearing loss.

A similar trend was observed in the smoking group. Participants with vertigo had higher mean hearing thresholds in both the good ear (Mean=28.19±9.63) and the bad ear (Mean=34.12±10.17) compared to those without vertigo (Good ear Mean=19.94±9.27; Bad ear Mean=22.12±9.66). These differences were also statistically significant (Good ear: $t(215)=-5.75$, $p < 0.001$; Bad ear: $t(215)=-7.98$, $p < 0.001$).

A Two-Way ANOVA was conducted to examine the effects of smoking and tinnitus on Bad ear hearing thresholds (PTA). Significant main effects were observed for both tinnitus, $F(1.438)=376.68$, $p < 0.001$, $\eta^2=0.462$, and smoking, $F(1.438)=12.93$, $p < 0.001$, $\eta^2=0.029$, indicating higher PTA values in individuals with tinnitus and in smokers. It was observed that smoking had a significant main effect on bad ear PTA values, $F(1.439)=24.59$, $p < 0.001$, $\eta^2=0.053$. This finding indicates that the mean bad ear hearing thresholds of smokers were significantly higher than those of non-smokers, thus showing that smoking increases hearing loss. Neither the interaction between smoking and tinnitus, $F(1.438)=0.02$, $p=0.892$, $\eta^2 < 0.001$, nor the interaction between smoking and vertigo, $F(1.437)=1.88$, $p=0.171$, $\eta^2=0.004$, was significant. These results suggest that tinnitus and smoking independently elevate Bad ear hearing thresholds, while their interactions do not significantly affect hearing.

Smoking Effect on the Inner Ear

Recent evidence indicates that cigarette smoke contains more than 4,000 distinct chemical compounds, many of which are toxic to the auditory system (Istrate et al., 2021; Hoffmann & Hoffmann, 1997). These compounds have been shown to adversely affect cochlear homeostasis through multiple mechanisms. Smoking can impair cochlear perfusion by inducing peripheral vascular alterations such as increased blood viscosity and reduced oxygen transport capacity, ultimately leading to hypoxia within the cochlea. Nicotine, as a potent vasoconstrictor, further compromises the delicate cochlear microcirculation, thereby promoting ischemic injury and contributing to sensorineural hearing loss that predominantly affects higher frequencies (Kazempour et al., 2023; Zhao, Liang, Pan et al., 2021).

Cigarette smoke contains thousands of toxic chemicals that increase the risk of sensorineural hearing loss (SNHL), particularly at high frequencies (Kazempour et al., 2023; Rogha, Hashemi, Askari, Abtahi, Sepehrnejad, & Nilforoush, 2015). This damage primarily affects outer hair cells (OHCs) in the cochlea, critical for sound perception (Myszel & Skarżyński, 2024). Smoke-induced cochlear injury occurs via two main mechanisms: hypoxia from carbon monoxide and vasoconstriction from nicotine, reducing cochlear blood flow (Dawes et al., 2014; Gates, Cobb, D'Agostino, & Wolf, 1993; Lin, Krall, Westerberg, Chadha, & Chau, 2012), and oxidative stress through reactive oxygen species, triggering OHC apoptosis (Seo et al., 2023; Zhao et al., 2021; Gill, Dowker-Key, Hedrick, & Bettaieb, 2024). Additionally, tobacco smoke may indirectly contribute to conductive hearing loss via Eustachian tube dysfunction and middle ear mucosal edema (Pezzoli, Lofaro, Oliva et al., 2017).

Atherosclerotic Cochlear Changes Due to Smoking

The cochlea relies on a single terminal artery, making it highly vulnerable to systemic vascular pathology (Kazempour et al., 2023; Pezzoli et al., 2017). Cigarette smoking, a well-established risk factor for cardiovascular disease and atherosclerosis, impairs cochlear perfusion through multiple mechanisms. Nicotine induces vasoconstriction, while elevated carboxyhemoglobin levels reduce oxygen delivery, together causing chronic cochlear hypoxia (Hoffmann & Hoffmann, 1997; Daiber, Kuntic, Oelze, Hahad, & Munzel, 2023; Sendesen, Demirtas, Turkyilmaz, & Sennaroglu, 2023). Endothelial dysfunction and plaque formation further compromise microcirculation, providing a pathophysiological basis for audiometric deficits. Epidemiological studies, including the Framingham and ARIC studies, confirm the link between smoking, systemic vascular disease, and hearing impairment (Garcia Morales et al., 2022; Dawes et al., 2014; Gates et al., 1993). In this study, the clinically significant PTA threshold shift in smokers supports the role of smoking-induced vascular changes in high-frequency sensorineural hearing loss.

Reactive Oxygen Species and Cochlear Damage from Smoking

Beyond ischemic damage from vascular alterations, oxidative stress represents a second key mechanism contributing to sensorineural hearing loss in smokers (Lin et al., 2012). Cigarette smoke contains numerous free radicals and induces reactive oxygen species (ROS) production by disrupting cellular antioxidant systems (Seo et al., 2023). Cochlear outer hair cells (OHCs) and supporting cells are highly metabolically active and particularly vulnerable to ROS-induced damage (Myszel & Skarżyński, 2024; Zhao et al., 2021). Oxidative injury to mitochondrial DNA and proteins activates the p53-mediated apoptotic pathway, leading to irreversible OHC loss, especially in the basal cochlea responsible for high-frequency hearing (Rogha et al., 2015; Lan, Lederman, Eng, Shoshtari, Saleem, Malhotra, & Singhal, 2016; Zhang L, Gao Y, Zhang R, Sun F et al., 2020). Our 5.34 dB HL difference observed in this

study, predominantly at higher frequencies, aligns with a dose-dependent, ROS-driven apoptotic mechanism, independent of macrovascular changes.

Eustachian Tube Dysfunction and Hearing Loss Related to Smoking

In addition to cochlear damage, smoking can contribute to middle ear pathology through Eustachian Tube Dysfunction (ETD) (Bhatt & Upreti, 2025; Pezzoli et al., 2017). Smoke-related toxins induce chronic inflammation and mucosal swelling in the nasopharynx and Eustachian tube lining, impairing mucociliary clearance (Monsell, 2019; Kumar, Gulati, Singhal, Hasan, & Khan, 2013). This functional compromise is reflected in markedly reduced tubal performance among smokers 59%, resulting in persistent negative middle ear pressure and adding a conductive component to hearing loss (Pezzoli et al., 2017). Therefore, smoke-related auditory impairment involves a dual mechanism, affecting both cochlear microcirculation and middle ear ventilation.

DISCUSSION

This study clearly demonstrated the negative impact of smoking on auditory and vestibular health within our cohort of 443 participants. The analyses revealed that the prevalence of tinnitus (42.2% vs. 26.8%) and vertigo (26.7% vs. 18.3%) was significantly higher in smokers. As the most crucial finding, smoking status was found to have a statistically significant main effect on bad ear PTA values, $F(1,439)=24.59$, $p<0.001$, $\eta^2=0.053$ The considerably high bad ear PTA mean (34.12 dB) observed in individuals who smoke and experience vertigo suggests that the combination of smoking and vertigo exacerbates hearing loss. Although the direction is similar in both groups, the higher difference in smokers suggests that smoking may aggravate hearing loss due to its oxidative stress and vascular effects on the vestibular and cochlear systems.

Lee et al., in a large Korean cohort, concluded that smoking is an independent cause of hearing loss even in young adults, highlighting the critical need for preventive interventions (Lee, Chang, Shin, & Ryu, 2021). Supporting this causality, the comprehensive review by Myszel and Skarżyński (2024) confirmed that both active and passive smoking are linked to sensorineural hearing loss, predominantly in the speech and high-frequency ranges, with a clear dose-response effect (Myszel & Skarżyński, 2024). Our results are consistent with these observations and further strengthen the hypothesis that cigarette smoke exerts a cumulative ototoxic effect over time.

The specific nature of the damage identified in our patients—predominantly high-frequency sensorineural loss—is corroborated by studies emphasizing cochlear vulnerability. Pezzoli et al. (2017) reported that tobacco impairs hearing primarily at higher frequencies and also increases the prevalence of Eustachian Tube Dysfunction in smokers, suggesting an extended impact beyond the cochlea (Pezzoli et al., 2017). Similarly, Sendesen et al. (2023) showed that the earliest effects of smoking manifest in extended high-frequency regions up to 20 kHz, corresponding to the basal cochlea (Sendesen et al., 2023). Mechanistically, Istrate et al. (2021) demonstrated that cigarette smoking compromises antioxidant defense and tissue metabolism, providing biological plausibility for the oxidative and microvascular mechanisms underlying our observed threshold shifts (Istrate et al., 2021).

While the overall evidence favors a causal link, some heterogeneity remains among studies. Huang et al. (2025), analyzing NHANES data, reported a significant association only in middle-aged males, implying that hormonal or lifestyle factors may modify susceptibility (Huang et al., 2025). Our findings, however, indicate that the detrimental auditory effects of smoking occur independent of gender**,** as no significant interaction between smoking and sex was observed in our analysis.

Taken together, these results highlight that smoking may contribute not only to cochlear hypoxia and oxidative stress but also to vestibular dysfunction, as reflected by the higher vertigo prevalence among smokers. The combination of increased hearing thresholds, tinnitus, and vertigo supports the multifactorial ototoxic impact of tobacco exposure.

Despite its strengths, this study has inherent limitations due to its retrospective design. Factors such as the absence of dose–year quantification and unmeasured occupational or environmental noise exposure could not be fully controlled, potentially influencing the strength of the observed associations. Future studies with larger cohorts, objective noise-exposure data, and prospective follow-up are warranted to confirm and expand upon these findings.

CONCLUSION

Smoking has a statistically and clinically significant adverse effect on auditory function, confirming an association with both elevated hearing thresholds and a greater prevalence of tinnitus and vertigo. This suggests that smoking acts as a multifactorial ototoxic risk factor, likely accelerating cochlear damage through its oxidative stress and vascular effects. These findings underscore the critical need for smoking cessation interventions to prevent progressive auditory and vestibular dysfunction. Future large-scale, prospective, and mechanistic studies are warranted to further elucidate the pathways through which smoking impacts auditory and vestibular health.

Data Availability Statement

The datasets generated during and/or analysed during the current study are not publicly available due to patient privacy restrictions but are available from the corresponding author on reasonable request.

Conflict of Interest

The authors declare that they have no conflict of interest.

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REFERENCES

Bener, A., Erdogan, A., & Griffiths, M. D. (2024). The impact of cigarette smoking, water-pipe use on hearing loss/hearing impairment: a cross-sectional study. *Asian Pac J Cancer Prev, 25*, 109–114. [CrossRef]

Bhatt, A. T., & Upreti, G. (2025). Association of smoking with hearing loss—a hospital based retrospective case-control study. *Egypt J Otolaryngol, 41*. [CrossRef]

Capaccio, P., Ottaviani, F., Cuccarini, V., Bottero, A., Schindler, A., Cesana, B. M., …, & Pignataro, L. (2007). Genetic and acquired prothrombotic risk factors and sudden hearing loss. *Laryngoscope, 117*, 547–551. [CrossRef]

Chau, J. K., Lin, J. R. J., Atashband, S., Irvine, R. A., & Westerberg, B. D. (2010). Systematic review of the evidence for the etiology of adult sudden sensorineural hearing loss. *Laryngoscope, 120*, 1011–1021. [CrossRef]

Cruickshanks, K. J., Klein, R., Klein, B. E., Wiley, T. L., Nondahl, D. M., & Tweed, T. S. (1998). Cigarette smoking and hearing loss the epidemiology of hearing loss study. *JAMA, 279*, 1715–1719. [CrossRef]

Daiber, A., Kuntic, M., Oelze, M., Hahad, O., & Munzel, T. (2023). E-cigarette effects on vascular function in animals and humans. *Pflugers Arch, 475*, 783–796. [CrossRef]

Dawes, P., Cruickshanks, K. J., Moore, D. R., Edmondson-Jones, M., McCormack, A., Fortnum, H., & Munro, K. J. (2014). Cigarette smoking, passive smoking, alcohol consumption, and hearing loss. *J Assoc Res Otolaryngol, 15*, 663–674. [CrossRef]

de Oliveira Penido, N., Ramos, H. V. L., Barros, F. A., Cruz, O. L. M., & Toledo, R. N. (2005). Clinical, etiological and progression factors of hearing in sudden deafness. *Rev Bras Otorrinolaringol, 71*, 633–638. [CrossRef]

Einer, H., Tengborn, L., Axelsson, A., & Edstrom, S. (1994). Sudden sensorineural hearing loss and hemostatic mechanisms [internet]. *Arch Otolaryngol Head Neck Surg, 120*, 536–540. [CrossRef]

Field, A. (2013). Factorial ANOVA. Two-way independent ANOVA. In *Discovering Statistics Using IBM SPSS Statistics* (4th ed., pp. 457–472). Sage Publications.

Garcia Morales, E. E., Ting, J., Gross, A. L., Betz, J. F., Jiang, K., Du, S., …, & Deal, J. A. (2022). Association of cigarette smoking patterns over 30 years with audiometric hearing impairment and speech-in-noise perception: the Atherosclerosis Risk in Communities Study. *JAMA Otolaryngol Head Neck Surg, 148*, 243–521. [CrossRef]

Gates, G. A., Cobb, J. L., D'Agostino, R. B., & Wolf, P. A. (1993). The relation of hearing in the elderly to the presence of cardiovascular disease and cardiovascular risk factors. *Arch Otolaryngol Head Neck Surg, 119,* 156–161. [CrossRef]

Gill, N. B., Dowker-Key, P. D., Hedrick, M., & Bettaieb, A. (2024). Unveiling the role of oxidative stress in cochlear hair cell death: prospective phytochemical therapeutics against sensorineural hearing loss. *Int J Mol Sci, 25*, 4272. [CrossRef]

Hoffmann, D., & Hoffmann, I. (1997). The changing cigarette, 1950–1995. *J Toxicol Environ Health, 50*, 307–364. [CrossRef]

Huang, H., Zhou, W., Chen, K., Du, Y., Lin, W., & Ye, Y. (2025). Association between cigarette smoking and hearing loss: a cross-sectional study from the NHANES database. *Tob Induc Dis, 23*. [CrossRef]

Istrate, M., Hasbei-Popa, M., Iliescu, D. A., Ghita, A. C., & Ghita, A. M. (2021). Effects of cigarette smoking on sensorineural hearing impairment and age-related macular degeneration. *Tob Prev Cessat, 7*, 55. [CrossRef]

Kazempour, M., Tajvar, A., & Heirani, A. (2023). Review of studies on the relationship between cigarette smoking and hearing loss and the related factors. *Tobacco Health, 2*, 64–69. [CrossRef]

Kline, R. B. (2016). *Principles and Practice of Structural Equation Modeling* (4th ed., pp. 73–75). The Guilford Press.

Kumar, A., Gulati, R., Singhal, S., Hasan, A., & Khan, A. (2013). The effect of smoking on the hearing status –a hospital based study. *J Clin Diagn Res,* 7, 210–214. [CrossRef]

Lan, X., Lederman, R., Eng, J. M., Shoshtari, S. S. M., Saleem, M. A., Malhotra, A., & Singhal, P. C. (2016). Nicotine induces podocyte apoptosis through increasing oxidative stress. *PLoS One, 11,* e0167071.

Lee, W., Chang, Y., Shin, H., & Ryu, S. (2021). Self-reported and cotinine-verified smoking and increased risk of incident hearing loss. *Sci Rep, 11,* 8103. [CrossRef]

Lin, B. M., Wang, M., Stankovic, K. M., Eavey, R., Mc,Kenna, M. J., Curhan, G. C., & Curhan, S. G. (2020). Cigarette smoking, smoking cessation, and risk of hearing loss in women. *Am J Med,* 133, 1180–1186. [CrossRef]

Lin, R. J., Krall, R., Westerberg, B. D., Chadha, N. K., & Chau, J. K. (2012). Systematic review and meta-analysis of the risk factors for sudden sensorineural hearing loss in adults. *Laryngoscope, 122*, 624–635. [CrossRef]

Monsell, E. M. (2019). Regarding evaluation and treatment of pulsatile tinnitus associated with sigmoid sinus wall anomalies. *Otology/Neurotology, 129*, E127. [CrossRef]

Myszel, K., & Skarżyński, P. H. (2024). Influence of smoking on human hearing – literature review. *Cent Eur J Public Health, 32*, 251–255. [CrossRef]

Pezzoli, M., Lofaro, D., Oliva, A., Orione, M., Cupi, D., Albera, A., …, & Albera, R. (2017). Effects of smoking on eustachian tube and hearing. *Int Tinnitus J, 21*, 98–103. [CrossRef]

Rogha, M., Hashemi, M., Askari, N., Abtahi, S. H., Sepehrnejad, M., & Nilforoush, M. H. (2015). Cigarette smoking effect on human cochlea responses. *Adv Biomed Res, 4*, 148.

Sendesen, E., Demirtas, B., Turkyilmaz, M. D., & Sennaroglu, G. (2023). The effect of smoking on hearing: the invisible part of the iceberg. *H.U. Faculty of Health Sci J, 10*, 259–273. [CrossRef]

Seo YS, Park JM, Kim JH, & Lee MY. (2023). Cigarette smoke-induced reactive oxygen species formation: a concise review. *Antioxidants (Basel), 12*, 1732. [CrossRef]

Tabachnick, B. G., & Fidell, L. S. (2019). *Using Multivariate Statistics* (7th ed., pp. 192–200). Pearson Education.

West, S. G., Finch, J. F., & Curran, P. J. (1995). Structural equation models with nonnormal variables: Problems and remedies. In R. H. Hoyle (Ed.), Structural Equation Modeling: concepts, issues, and applications (pp. 56–75). Sage Publications.

World Health Organization (WHO). (2021). *World Report on Hearing*. Geneva.

Zhang L, Gao Y, Zhang R, Sun F, Cheng C, Qian F, …, & Liu, D. (2020). THOC1 deficiency leads to late-onset nonsyndromic hearing loss through p53-mediated hair cell apoptosis 2019. [CrossRef]

Zhao, Y., Liang, Y., Pan, C., Tang, X., Sun, Y., Xu, C, …, & Sun, J. (2021). Nicotine induced ototoxicity in rat cochlear organotypic cultures. *Transl Neurosci, 12*, 407–414. [CrossRef]